Safety Data Sheet

Acrylonitrile

Division of Safety
National Institutes
of Health

WARNING!

THIS COMPOUND IS ABSORBED THROUGH THE SKIN AND RESPIRATORY AND INTESTINAL TRACTS. IT IS TOXIC, CARCINOGENIC, MUTAGENIC, AND TERATOGENIC. IT IS FLAMMABLE AND EXPLOSIVE. AVOID FORMATION AND BREATHING OF AEROSOLS OR VAPORS. IT IS IRRITATING TO EYES AND SKIN.

LABORATORY OPERATIONS SHOULD BE CONDUCTED IN A FUME HOOD, GLOVE BOX, OR VENTILATED CABINET.

AVOID SKIN CONTACT: IF EXPOSED, WASH WITH SOAP AND WATER.

FOR EYE EXPOSURE, IRRIGATE IMMEDIATELY WITH LARGE AMOUNTS OF WATER. FOR INGESTION, APPLY LAVAGE WITH SOLUTION OF 52 GRAMS OF SODIUM THIOSULFATE IN ONE LITER OF WATER. INDUCE VOMITING. FOR INHALATION, REMOVE VICTIM PROMPTLY TO CLEAN AIR. MAKE VICTIM INHALE FUMES FROM AMYL NITRITE AMPOULES. ADMINISTER RESCUE BREATHING IF NECESSARY. REFER TO PHYSICIAN.

IN CASE OF LABORATORY SPILL, WEAR PROTECTIVE CLOTHING DURING CLEANUP. AVOID SKIN CONTACT AND BREATHING OF AEROSOLS OR VAPORS. WASH DOWN AREA WITH SOAP AND WATER. DISPOSE OF WASTE SOLUTIONS APPROPRIATELY.

A. Background

Acrylonitrile (AN) is a colorless, volatile, flammable, and explosive liquid. It is toxic, carcinogenic, mutagenic, and teratogenic. Toxic effects in humans are slight jaundice, gastritis, respiratory difficulties, fatigue, and headache. AN is an important industrial chemical; because of its ease of polymerization and co-polymerization with

issued: 11/82

Prepared by the Environmental Control and Research Program

other materials, it is used extensively in the production of acrylic fibers such as Orlon, plastics, surface coatings, and adhesives. It is also used as a grain fumigant and in organic synthesis for cyanoethylations.

The permissible exposure limit to AN is 2 ppm as an 8-hour time-weighted average (ACGIH, 1986).

Chemical and Physical Data

1. Chemical Abstract No.: 107-13-1

2. Synonyms:

ACN	Carbacryl
VCN	Fumigrain
ENT 54	Cyanoethylene
TL 314	Vinyl cyanide
Ventox	Acrylonitrile monomer
Acrylon	2-Propenenitrile (9CI)

3. Molecular formula: C_3H_3N structure: $CH_2 = CHCN$

   weight: 53.07

4. Density: Liquid, 0.8060 g/cm^3 (20°C/4°C); vapor (air = 1), 1.83.

5. Absorption spectroscopy: UV λ (log ε) = 203 (3.79). Data for infrared, Raman, NMR, and mass spectra have been tabulated (Grasselli and Ritchey, 1975). The infrared absorption spectrum has been published (Pouchert, 1970; Jacobs and Syrjala, 1978).

6. Volatility: Vapor pressure = 100 mm Hg at 22.8°C. (For vapor pressures at other temperatures, see p. D-204 in Weast, 1979.)

7. Solubility: Soluble in water (7.35 parts AN dissolve in 100 part water at 20°C); soluble in acetone and benzene; miscible with ethanol and ether.

8. Description: Clear, colorless liquid with characteristic odor resembling that of peach seeds.

9. Boiling point: 77.5-79°C.
   Melting point: -83.5°C.

10. Stability: Relatively stable when stored in the freezing compartment of an explosion-safe refrigerator. Flammable and explosive. Polymerizes spontaneously, particularly on exposure to heat and in the presence of strong oxidizers or alkali. (Note: most "pure" preparations of AN contain a polymerization inhibitor such as 35-40 ppm of hydroquinone monomethyl ether, which can be removed by distillation.) Stock solutions in hexane are stable when refrigerated as above; dilute solutions are stable for a week in the refrigerator, even in absence of the polymerization inhibitor (Gagnon and Posner, 1979).

11. Chemical reactivity: Hydrolyzed by acid through acrylamide to acrylic acid. The double bond can be hydrogenated and undergoes Diels-Alder reactions. Undergoes usual reactions of the nitrile group. Decomposes in the presence of copper and copper alloys (summarized in Kirk-Othmer, 1978).

12. Flash point: -1°C (open cup).

13. Autoignition temperature: 481°C.

14. Explosive limits in air: 3-17%.

Fire, Explosion, and Reactivity Hazard Data

1. Use foam, carbon dioxide, or dry chemical fire extinguishers. Fire-fighting personnel should wear air-supplied respirators with full-face masks.

2. AN is flammable and its vapors in air can produce explosive mixtures.

3. Hazardous decomposition products at high temperatures include hydrogen cyanide, nitrogen oxides, and carbon monoxide.

4. Other conditions contributing to instability: strong oxidizers and alkalis will promote explosive polymerization. Avoid contact with copper and its alloys, ammonia, and amines. Heat may cause containers to explode.

5. Do not expose to sparks or open flames. Use nonspark tools and equipment. Store in a refrigerator designed to permit the safe storage of flammable solvents.

Operational Procedures

The NIH Guidelines for the Laboratory Use of Chemical Carcinogens describe operational practices to be followed when potentially carcinogenic chemicals are used in NIH laboratories. The NIH Guidelines should be consulted to identify the proper use conditions required and specific controls to be implemented during normal and complex operations or manipulations involving AN.

It should be emphasized that this data sheet and the NIH Guidelines are intended as starting points for the implementation of good laboratory practices when using this compound. The practices and procedures described in the following sections pertain to the National Institutes of Health and may not be universally applicable to other institutions. Administrators and/or researchers at other institutions should modify the following items as needed to reflect their individual management system and current occupational and environmental regulations.

AN penetrates various glove materials (Sansone and Tewari, 1978). This factor should be taken into account when handling AN.

1. Chemical inactivation: No validated method reported.

2. Decontamination: Turn off equipment that could be affected by AN or the materials used for cleanup. If there is any uncertainty regarding the procedures to be followed for decontamination, call the NIH Fire Department (dial 116) for assistance. Wash surfaces with copious quantities of water. Glassware should be kept in the hood until evaporation is deemed to be complete and then rinsed with alcoholic NaOH; the usual washing procedures should follow. (Personnel performing these operations should wear rubber gloves.)

3. Disposal: No waste streams containing AN shall be disposed of in sinks or general refuse. Surplus AN or chemical waste streams contaminated with AN shall be handled as hazardous chemical waste and disposed of in accordance with the NIH chemical waste disposal system. Nonchemical waste (e.g., animal carcasses and bedding) containing AN shall be handled and packaged for incineration in accordance with the NIH medical-pathological waste disposal system. Potentially infectious waste (e.g., tissue cultures) containing AN shall be packaged for incineration, as above. Burnable waste (e.g., absorbent bench top liners) minimally contaminated with AN shall be handled as potentially infectious waste and packaged for incineration, as above. Absorbent materials (e.g., associated with spill cleanup) grossly contaminated shall be handled in accordance with the chemical waste disposal system. Radioactive waste containing AN shall be handled in accordance with the NIH radioactive waste disposal system.

4. Storage: Store in sealed ampoules or in screw-capped bottles (or vials) with Teflon cap liners in the freezing compartment of an explosion-safe refrigerator. Stock solutions in organic solvents (such as hexane) may be similarly stored. More dilute solutions are stable for a week when stored in a regular refrigerator. Do not store in metal containers.

Monitoring and Measurement Procedures Including Direct Field Measurements and Sampling for Subsequent Laboratory Analysis

1. Sampling: The officially (NIOSH) recommended method for air sampling is adsorption on charcoal and desorption with methanol (NIOSH, 1978). In a later report, Gagnon and Posner (1979) used 2% acetone in carbon disulfide for desorption of charcoal. More recently it has been argued that at the low monitoring level required by the latest OSHA standar recovery from charcoal is unsatisfactory, particularly at high humidity levels (Campbell and Moore, 1979). A variety of porous polymer traps have been used such as Porapak N (Campbell and Moore, 1979; Russell, 1975) or Tenax GC (Parsons and Mitzner, 1975), from which AN can be desorbed thermally. The validity of the above objection to the use of charcoal has been disputed (Gagnon and Posner, 1979).

2. Analysis: Older methods include polarography for macro amou (e.g., 0.5% in water) (Bird and Hale, 1952) and colorimetry, either of excess iodine remaining after reaction of AN with lauryl mercaptan (Haslam and Newlands, 1955) (useful range, 0-150 mg/m^3 in air) or of the yellow reaction product with pyridine in the presence of a basic hypochlorite (Hall and Stevens, 1977). Although the latter method is useful for aqueous solutions, the likely presence of cyanide causes strong interference.

   All newer methods are based on gas chromatography, usually with a flame ionization detector (Parsons and Mitzner, 1975; Russell, 1975; Campbell and Moore, 1979). Greater sensitivi (detection limit, 10 pg) and the possibility of direct air analysis (without concentration on charcoal or polymer) down to 10 ppb have been claimed for GC with a nitrogen-selective detector (Marano et al., 1978). A commercial portable infrared gas analyzer that monitors AN in air at the 0.2 ppm level, as well as other air pollutants, has been described (Jacobs and Syrjala, 1978).

Biological Effects (Animal and Human)

1. Absorption: AN is absorbed and produces toxic effects by in lation, by ingestion, and through the skin. It is a potent eye irritant but it is not known whether systemic effects are produced via this route. It is also toxic by the transplacental route.

2. Distribution: No specific data are available.

3. Metabolism and excretion: It was assumed by early investigators that the actions of AN were those of a typical nitril and could have been ascribed to the liberation of cyanide ion (Dudley and Neal, 1942); it was, however, noted even then that no free cyanide was detected in the blood of animals after exposure or in blood or tissues after death.

Later work has led to the conclusion that, at best, only a small, if any, contribution to toxicity is made by the liberation of cyanide (apparently depending on animal species used) on the basis of symptomatology and physiological, biochemical, and toxicological effects (Ghiringelli, 1954, 1956; Paulet and Desnos, 1961). It is generally believed now that the main metabolism is an oxidative one through a reactive species, probably the epoxide across the double bond, which reacts with tissue constituents. Nevertheless, liberation of cyanide plays a small though variable role as shown by the excretion of cyanide and thiocyanate in the urine of some species (including man) and the therapeutic effect of nitrites and thiosulfate in the same animal species. In vitro, AN has been found to cyanoethylate unusual nucleosides (pseudouridine, inosine, 4-thiouridine), which are found only in transfer ribonucleic acid, and this may have a bearing on its mechanism of action (Ofengard, 1967; Ofengard and Henes, 1969).

Toxic effects: The acute LD50s in the mouse (in mg/kg) are 27 (oral), 44 (intraperitoneal), and 35 (subcutaneous); in the rat, acute LD50s are 78, 100, and 80 by the same routes. Figures for rabbits and guinea pigs are similar. Dermal toxicity through abraded skin is 0.28 and 0.84 ml/kg in the rabbit and guinea pig, respectively. Toxic effects are those of a severe eye irritant (lacrimation, corneal reddening or lesions), nasal irritation, and actions on the central nervous system (fasciculation, tetany, convulsions, and paralysis). Prolonged skin exposure may produce blisters after several hours, resembling those due to thermal burns. Symptoms in man following accidental exposures have been described (Wilson, 1944) and include nausea, vomiting, weakness, fatigue, and diarrhea. In rats, intravenous administration of AN resulted in bilateral hemorrhages in the adrenal cortex ("adrenal apoplexy") and in the profound lowering of liver, adrenal, brain, lung, and kidney glutathione levels, presumably due to reaction of an "active intermediate" (epoxide?) of AN with tissue sulfhydryl groups (Szabo et al., 1977).

Carcinogenic effects: Ongoing studies, quoted in preliminary publications, indicate the development of tumors, incuding carcinomas, in rats after oral administration or inhalation of AN. A preliminary epidemiological study by Dupont shows an increased cancer rate among workers in an AN manufacturing plant (NIOSH, 1978). AN has been identified by an IARC Working Group as being probably carcinogenic for humans (Subgroup 2B) (Althouse et al., 1980).

Mutagenic and teratogenic effects: AN is mutagenic in the Ames test in the presence of a liver microsome activating system and is teratogenic in pregnant rats when administered either by gavage or inhalation.

Emergency Treatment

1. Skin and eye exposure: For skin exposure, remove contaminated clothing and wash skin with soap and water. For eye exposure, irrigate immediatey with copious quantities of running water for at least 15 minutes.

2. Ingestion: Apply lavage with a solution of 52 grams of sodium thiosulfate in one liter of water. Induce vomiting.

3. Inhalation: Remove victim promptly to clean air. Cause patient to inhale the vapor of one amyl nitrate ampoule in a handkerchief. Repeat inhalation procedure several times at about 15-second intervals.

4. Refer to physician immediately. Consider treatment for cyanide poisoning (Wilson and McCormick, 1949; Hamilton and Hardy, 1974; Vogel et al., 1981; Rumack, 1982).

References

ACGIH. 1986. Threshold limit values and biological exposure indices for 1986-1987. American Conference of Governmental Industrial Hygienists, Cincinnati, OH.

Althouse, R., J. Huff, L. Tomatis, and J. Wilbourn. 1980. An evaluation of chemicals and industrial processes associated with cancer in humans based on human and animal data: IARC Monograph Volumes 1 to 20. Cancer Res 40:1-12.

Bird, W.L., and C.H. Hale. 1952. Polarographic determination of acrylonitrile. Anal Chem 24:586-587.

Campbell, D.N., and R.H. Moore. 1979. The quantitative determination of acrylonitrile, acrolein, acetonitrile and acetone. Am Ind Hyg Assoc J 40:904-909.

Dudley, H.C., and P.A. Neal. 1942. Toxicology of acrylonitrile (vinyl cyanide). I. A study of the acute toxicity. J Ind Hyg Toxicol 24:27-36.

Gagnon, Y.T., and J.C. Posner. 1979. Recovery of acrylonitrile from charcoal tubes at low levels. Am Ind Hyg Assoc J 40:923-925.

Ghiringhelli, L. 1954. Acrylonitrile: Toxicity and mechanism of action. Med Lav 45:305-312.

Ghiringhelli, L. 1956. Comparative study of the toxicity of some nitriles and amides. Med Lav 47:192-199.

Grasselli, J.G., and W.M. Ritchey, eds. 1975. CRC Atlas of Spectral Data and Physical Constants for Organic Compounds. 2nd ed., Vol. II. CRC Press, Cleveland, OH.

Hall, M.E., and J.W. Stevens, Jr. 1977. Spectrophotometric determination of acrylonitrile. Anal Chem 49:2277-2280.

Hamilton, A., and H.L. Hardy. 1974. Industrial Toxicology, 3rd ed. Publishing Sciences Group, Inc., Acton, MA.

Haslam, J., and G. Newlands. 1955. Determination of acrylonitrile in air. Analyst 80:50-53.

Jacobs, H.W., and R.H. Syrjala. 1978. The use of infrared analyzers for monitoring acrylonitrile. Am Ind Hyg Assoc J 39:161-165.

Kirk, R.E., and Othmer, D.F. (eds.) 1978. Pages 414-426 in Encyclopedia of Chemical Technology, 3rd ed, Vol. 1. Wiley, New York, NY.

Marano, R.S., S.P. Levine, and T.M. Harvey. 1978. Trace determination of subnanogram amounts of acrylonitrile in complex matrices by gas chromatography with a nitrogen-selective detector. Anal Chem 50:1948-1950.

NIOSH, National Institute for Occupational Safety and Health. 1978. Recommended Standard for Occupational Exposure to Acrylonitrile DHEW (NIOSH) Publ. No. 78-116. U.S. Department of Health, Education and Welfare, Washington, DC.

Ofengard, J. 1967. The function of pseudouridylic acid in transfer ribonucleic acid. J Biol Chem 242:5034-5045.

Ofengard, J., and C. Henes. 1969. The function of pseudouridylic acid in transfer ribonucleic acid. II. J Biol Chem 244:6241-6253.

Parsons, J.S., and S. Mitzner. 1975. Gas chromatographic method for concentration and analysis of traces of industrial organic pollutants in environmental air and stacks. Environ Sci Technol 9:1053-1058.

Paulet, G., and J. Desnos. 1961. Acrylonitrile: Toxicity, mechanism of therapeutic action. Arch Int Pharmacodyn Ther 131:54-83.

Pouchert, C.J. 1970. Page 333H in The Aldrich Library of Infrared Spectra. Aldrich Chemical Company, Milwaukee, WI.

Rumack, B.H. (ed.). 1982. Poisindex. Microfiche Edition, Denver, Colorado, Micromedex, Inc., in association with the National Center for Poison Information.

Russell, J.W. 1975. Analysis of air pollutants using sampling tubes and gas chromatography. Environ Sci Technol 9:1175-1178.

Sansone, E.B., and Y.B. Tewari. 1978. Penetration of protective clothing materials by 1,2-dibromo-3-chloropropane, ethylene dibromide and acrylonitrile. Am Ind Hyg Assoc J 39:921-922.

Szabo, S., K.A. Bailey, P.J. Boor, and R.J. Jaeger. 1977. Acrylonitrile and tissue glutathione: Differential effect of acute and chronic interactions. Biochem Biophys Res Commun 79:32-37.

Vogel, S.N., T.R. Sultan, and R.P. Ten Eyck. 1981. Cyanide poisoning. Clin Toxicol 18:367-383.

Weast, R.C. (ed.). 1979. Handbook of Chemistry and Physics, 60th ed., CRC Press, Inc., Boca Raton, FL.

Wilson, R.H. 1944. Health hazards encountered in the manufacture of synthetic rubber. JAMA 124:701-703.

Wilson, R.H., and W.E. McCormick. 1949. Acrylonitrile: Its physiology and toxicology. Indust Med 18:243-245.